The
VETERINARY RECORD
Founded by William Hunting, F.R.C.V.S., in 1888
7, MANSFIELD STREET, PORTLAND PLACE, LONDON, W.1. MUSEUM 6541
No. 46 NOVEMBER 12th, 1955 Vol. 67
Registered for Transmission as a Newspaper *Price 2s. post free*

The Use of Furazolidone in the Treatment of Infections of Day-old Chicks with *S. pullorum*, *S. gallinarum*, *S. typhi-murium* and *S. thompson*

BY
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Introduction

FURAZOLIDONE (N-(5-nitro-2-furfurylidene)-3-amino-2 oxazolidone), first synthesised by Stillman and Dodd of Easton Laboratories, U.S.A., and shown by Yurchenco, Yurchenco and Piepoli (1953) to have pronounced anti-bacterial action against gram-negative bacilli, including salmonella, has attracted attention in the U.S.A. and in this country chiefly perhaps as a potentially effective agent in the treatment and prevention of fowl typhoid.

Its use in fowl typhoid was first suggested by Harwood (1954) who found it highly effective in controlling a serious outbreak of this disease in turkeys in which there was a mortality of 100 per cent. in an untreated control pen. Successful results have also been described in U.S.A. by Grumbles, Wills & Boney (1954), Harwood & Stunz (1954), and Lucas (1955). In Great Britain, Williams Smith (1954a) reported that when 90 nine-week-old chicks experimentally infected with fowl typhoid were given furazolidone at a concentration of 0.02 per cent. in the mash, two died and only one of the 88 survivors developed agglutinins for *Salmonella gallinarum*. A full account of the chemotherapy of experimental fowl typhoid was later provided by the same worker (Williams Smith, 1955). Furazolidone was greatly superior to sulphonamides, dihydrostreptomycin, chloramphenicol, terramycin and aureomycin in the treatment of experimental fowl typhoid in one-day-old chicks and nine-week-old chickens. Williams Smith (1954b) showed that furazolidone was also effective against *S. pullorum* infection in chicks and concluded that the most satisfactory therapy was 0.04 per cent. of the drug in the mash continuously for 10 to 14 days.

Following a diagnosis of *Salmonella* infection in chicks, the disposal of survivors often presents a difficult problem. In the official schemes slaughter is usually recommended. Where there has been a heavy mortality an owner will usually readily consent to this procedure but in mild outbreaks where deaths are few there is generally unwillingness to comply; a compromise may be reached by retaining the chicks and disposing of them for table purposes at the earliest opportunity.

It is from the less severe outbreaks that proportionately larger numbers of carriers usually stem. It seemed that furazolidone, by controlling mortality and lowering the incidence of carriers, might be even more valuable in the treatment of salmonellosis of young stock than in fowl typhoid.

The present paper describes a series of experiments begun in the summer of 1954 to determine the value of the drug against experimental infections of day-old chicks with *S. pullorum*, *S. gallinarum*, *S. typhi-murium* and *S. thompson*. The results, using a concentration of 0.04 per cent. of the drug, in the mash, are in general agreement with those of Williams Smith (1954b); at the lower concentration (0.02 per cent.) they are less satisfactory than those described in his original paper (Williams Smith, 1954a). As the type of outbreaks produced closely approximates those occurring naturally, it is considered that the results obtained are likely to be valid under field conditions.

Material and Methods

Pure bred Light Sussex, White Wyandotte, Brown Leghorn, Rhode Island Red and Black Leghorn/Rhode Island Red cross day-old chicks were used. The test organisms were grown on agar from broth

cultures of the dried organism. Saline washings were standardised to Tube 6 Brown's scale. In Experiment 1 chicks were infected by introducing 0.5 ml. of the suspension into the crop by means of a pipette. This method of individual dosing was also used by Williams Smith (1954a). It is tedious and time-consuming and has little to recommend it as a means of infecting day-old chicks.

In experiments 2, 3 and 4, chicks were infected by the spray method devised by the writer nearly 25 years ago (Wilson, 1931). Although at that time it was commonly believed that ingestion of food and water contaminated by *S. pullorum* was chiefly responsible for natural outbreaks of bacillary white diarrhoea, difficulty was experienced in reproducing the disease experimentally in chicks which received drinking water to which washings of agar cultures of *S. pullorum* were added.

Results were uncertain; even in day-old chicks the mortality was never high and chicks over three days old were refractory as far as the production of disease with any appreciable mortality was concerned. As the disease is egg-borne, infection within the incubator by inhalation of germs on dust and fluff appeared more likely and it was found that by spraying a suspension of *S. pullorum* over healthy day-old chicks and returning them to the incubator to dry off, disease having a mortality of 100 per cent. could be set up. The death rate varied according to the proportion of chicks sprayed, *e.g.*, to produce an outbreak with an almost negligible mortality and a high proportion of carriers amongst the survivors it was sufficient to spray the down of the heads of two chicks and place them in an incubator with a group of healthy chicks. When equal amounts of *S. pullorum* suspension were administered to equivalent groups of chicks by spraying and adding to the drinking water, the mortality resulting from the former method was about four times greater than that from the latter.

As a result of these experiments, a standard procedure was adopted for infecting day-old chicks with any of the *Salmonella* group of organisms. Saline washings of nutrient agar cultures of the test organism standardised to Tube 6 Brown's scale are used at the rate of 10 ml. for 150 to 200 day-old chicks. During the process of spraying and for 30 minutes afterwards the chicks are held either in an incubator at 100° F. or in a large cardboard container in a warm room. They are then divided into random groups for experimentation.

Following spraying with *S. pullorum* and *S. gallinarum* a mortality rate of 100 per cent. may be expected. The course of the disease follows a standard pattern. Deaths begin on the fifth day and reach a peak on the sixth day with about the same number of deaths on the seventh day as on the fifth, *e.g.*, in three experiments involving groups of 45 to 48 chicks the daily death rates were, fifth day 9, 9, 10, sixth day 24, 23, 23, and seventh day 9, 8, and 11. With other members of the *Salmonella* group, the mortality is variable; strains appear to differ considerably in pathogenicity.

Apart from the ease of administration and regularity of infection, this method has the additional advantage that as it closely simulates the natural mode of dissemination of infection and produces outbreaks of disease similar to those occurring in the field, it is likely to provide a reliable indication of the practical value of any drug or other control measure proving successful experimentally.

Bacteriological Examination of Chicks

Autopsies were performed on all chicks which died or were destroyed during the experiments. Bacteriological procedures varied according to circumstances. In infections with *S. pullorum* and *S. gallinarum*, chicks from the untreated control groups invariably showed gross evidence of disease (enlarged liver and spleen, nodules in lungs and myocardium) and direct cultures from the livers on MacConkey medium were sufficient to isolate the organism. Chicks which died or were destroyed from all other groups were examined more minutely as it was essential to determine if they were carriers. The liver and lungs of each chick were removed aseptically, ground up in a Griffiths tube and added to 100 ml. of Selenite F medium (Leifson, 1936) in which the disodium salt was substituted for the two phosphate salts and the *p*H adjusted by N/1 HCl (Hobbs & Allison, 1945). After incubation at 37° C. for 20 hours, subcultures were made on MacConkey media which were examined for the presence of salmonella after 20 to 24 hours' incubation.

In the early experiments the entire intestinal tract of each chick was minced up and cultured in Selenite F. media and examined for salmonella by the method already described. In later experiments an emulsion of the intestinal contents in normal saline was substituted. Samples of faeces from the various groups were also examined during the course of the experiments but cloacal swabbing was not carried out. Previous experience has shown the limitations of the latter method.

Results

Experiment 1.

One hundred and fifty day-old chicks were infected by introducing a suspension of *S. gallinarum* directly into the crop as previously described, and divided into two random groups.

Two chicks, which inadvertently received some of the suspension into the trachea, died 48 hours later, with massive nodule formation of both lungs. Twenty-four hours after infection one group of chicks was given a commercial chick mash to which furazolidone was added to give a concentration of 0.02 per cent. Treatment was continued for five days. The other group received mash only. The results are shown in Table I (*opposite*).

Treatment was highly effective in controlling the disease. Losses in the treated pen during the first 21 days amounted to four chicks (5.3 per cent.), compared with 68 (90.6 per cent.) in the control pen. It is apparent that the drug was not completely bactericidal. Twenty days after treatment ceased a serious breakdown occurred; nine chicks died during

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FIG. 1.—Hearts of chicks infected with *S. pullorum* (Experiment 4).

17 — Treatment with Furazolidone.
3 and 32 — Treated with Sulphaquinoxaline.
29, 43, NN3 — Untreated controls.

FIG. 2.—Lungs of chicks infected with *S. pullorum* (Experiment 4).

Top — Treated with Furazolidone.
Middle — Treated with Sulphaquinoxaline.
Bottom — Untreated controls.

TABLE I

THE EFFECT OF TREATMENT WITH FURAZOLIDONE (0.02 PER CENT.) FOR FIVE DAYS STARTING 24 HOURS AFTER INFECTION OF DAY-OLD CHICKS WITH *S. gallinarum*.

Age of chicks (days)	Group 1 75 chicks 0·02 Furazolidone	Group 2 75 chicks Control
	Deaths	Deaths
1–7	2	47
8–14	1	19
15–21	1	2
22–28	7	0
29–35	6	0
36–42	3	0
Total deaths	20 (26·7%)	68 (90·6%)

the next four days and deaths continued sporadically for a fortnight. Later, all surviving birds were tested by the rapid whole blood test. Six were positive and from four of them *S. gallinarum* was isolated. The organism was also isolated from six birds which had given negative reactions to the test.

Experiment 2.

In some parts of the U.S.A. fowl typhoid presents a serious problem in young chicks. This is not so in this country where the disease in chicks has been rare. Until last year the writer had not come across any outbreak which appeared to be egg-borne, although bacteriological examination of large numbers of eggs laid by carriers, survivors of the naturally and experimentally produced disease had shown that many contained *S. gallinarum*. With the increased incidence of fowl typhoid in some parts of Great Britain it is probable that the disease in chicks arising from incubator infection will become more common. To test the efficiency of furazolidone against an experimentally produced incubator infection, chicks were infected by the spray method already described. An additional group of chicks was included to study the effect of delayed treatment. The results are shown in Table II.

TABLE II

THE EFFECT OF TREATMENT WITH FURAZOLIDONE (0·02 PER CENT.) FOR FIVE DAYS, BEGUN 24 AND 72 HOURS AFTER INFECTION OF TWO GROUPS OF DAY-OLD CHICKS WITH *S. gallinarum*

Age of chicks (days	Group 1 47 chicks Furazolidone 24 hours after infection	Group 2 47 chicks Furazolidone 72 hours after infection	Group 3 46 chicks Control
	Deaths	Deaths	Deaths
1–7	0	0	42
8–14	6	4	2
15–21	9	2	1
22-28	0	0	1
Total deaths	15 (31·9%)	6 (12·8%)	46 (100%)

The marked protective effect of furazolidone 0.02 per cent. is again clearly shown. During the period of treatment mortality was completely prevented, while it was almost 100 per cent. in the control group. As in the previous experiment a breakdown occurred after treatment had ceased, six days afterwards in brooder 1, and four days afterwards in brooder 2; in both cases 12 days after initial infection. It is not possible to draw any valid conclusion on the effect of the delayed administration of furazolidone, but the results confirm that a concentration of 0.02 per cent. is not bactericidal.

Three more deaths from fowl typhoid occurred in brooder 2, 36 days, 48 days and 113 days after initial infection. All surviving birds were negative to agglutination tests (rapid whole blood and tube methods) and *S. gallinarum* was isolated from one bird only.

Experiment 3.

This experiment was designed to test the efficiency of furazolidone against *S. typhi-murium* infection in chicks. In view of the results of the earlier experiments, the drug was fed at two levels, 0.02 per cent. and 0.04 per cent. and treatment which began 72 hours after infection was continued 12 days.

TABLE III

THE EFFECT OF TREATMENT WITH FURAZOLIDONE (0·02 AND 0·04 PER CENT.) FOR 12 DAYS STARTING 72 HOURS AFTER INFECTION OF DAY-OLD CHICKS WITH *S. typhi-murium*

Age of chicks	Group 1 34 chicks Furazolidone 0·02 per cent.	Group 2 32 chicks Furazolidone 0·04 per cent.	Group 3 33 chicks Control
	Deaths	Deaths	Deaths
1–7	2	1	3
8–14	0	0	1
15–21	0	0	0
22–28	0	1	0
Survivors	32	30	29
	S. typh.	*S. typh.*	*S. typh.*
Bacteriological Examination	+ 11 − 21	+ 4 − 26	+ 18 − 11

This experiment was only partially successful as a virulent outbreak of salmonellosis did not develop. This was not entirely unexpected as in the past it has proved difficult to induce a significant mortality in newly hatched chicks by artificial infection with *S. typhi-murium* even when the virulence of the organism has been increased by passage or the resistance of the chicks has been lowered by keeping at a reduced temperature (Wilson, 1945).

The number of chicks dying from salmonellosis is too small to allow significant deductions to be made. A death rate in the control group double that of the treated pen, however, suggests that the drug may be helpful in controlling outbreaks of the disease. The surviving chicks were destroyed, some at six weeks of age, the rest at eight weeks. The effect of treatment on the incidence of carriers is most striking. There were only four out of 30 in the group receiving 0.04 per cent. furazolidone, 11 out of 32 receiving 0.02 per cent. of the drug compared with 18 out of 29 in the control pen.

Experiment 4.

This experiment was designed to compare the effect of furazolidone (0.04 per cent. in the food for seven days), and sulphaquinoxaline (0.05 per cent. in the drinking water for five days), against artificial infection of day-old chicks with *S. pullorum*. Treatment with both drugs was begun 72 hours after infection. Results are shown in Table IV.

TABLE IV

THE EFFECT OF TREATMENT WITH FURAZOLIDONE (0·04 PER CENT.) AND SULPHAQUINOXALINE (0·05 PER CENT.) BEGUN 72 HOURS AFTER THE INFECTION OF 80 DAY-OLD CHICKS WITH *S. pullorum*.

Age of chicks (days)	Group 1 50 chicks Furazolidone 0·04 per cent. for 7 days	Group 2 49 chicks Sulphaquinoxaline 0·05 per cent. for 5 days	Group 3 49 chicks Control
	Deaths*	Deaths	Deaths
1–7	0	10	32
8–14	0	5	14
Intercurrent deaths	2	3	0
Survivors	48	31	3
S. pullorum –	1	31	3

* From pullorum disease.

The protective effect of furazolidone is apparent. None of the chicks in the treated pen ever showed evidence of illness, and the only two deaths which occurred were from accidental causes. *S. pullorum* was isolated from one of these chicks and from one of the 48 chicks destroyed immediately after treatment had ceased.

During the same period 45 out of 48 control chicks died. All had extensive lung lesions, and in many, necrotic nodules in the myocardium were also present. There were similar lesions in the three survivors destroyed at the end of the experiment.

Ten of the chicks receiving sulphaquinoxaline died from pullorum disease during treatment and five, 48 hours after treatment had stopped. There was gross evidence of the disease in every one of the 31 survivors from 30 of which *S. pullorum* was isolated by direct culture from the liver. The organism was isolated from the intestinal contents of 27 of the chicks. The presence of advanced disease in the survivors suggested that if the period of the experiment had been extended the death rate would have been very much greater.

Experiment 5.

The foregoing trials have shown the effectiveness of furazolidone under certain limited and well-defined experimental conditions. Its usefulness in the field would depend largely on its effect when treatment is delayed until the presence of disease is established, except perhaps on the relatively rare occasions when a previous history indicates the possibility of infection. The continuous feeding of a drug on the chance of chicks becoming infected is to be deprecated. This experiment was designed to create conditions simulating as closely as possible the circumstances which might arise in the field.

A group of 49 healthy day-old chicks was placed in a brooder near those containing the infected chicks of Experiment 4 in the hope that they might become infected. To ensure that any spread was completely natural, and not aided or hindered in any way, the attendant was left unaware of the nature of the experiment.

On the sixth day (a day after the first deaths in Experiment 4) there were six deaths from pullorum disease and treatment at 0.04 per cent. concentration was begun and continued for 10 days. Many of the chicks were obviously ill and four died the next day. A marked improvement in the appearance of the chicks took place and there were no further deaths for three days. During the remaining six days of treatment there were five single deaths.

Three more deaths occurred during the next three weeks when the experiment ended. Of the 28 survivors, three were undersized but the remainder appeared to be normal. *S. pullorum* was isolated from 10 (35.7 per cent.). The total death rate from pullorum disease, including those before treatment began was 18 (36.7 per cent.).

Experiment 6.

This experiment was to test the effect of furazolidone against infection of day-old chicks with *S. thompson*. After spraying, the chicks were divided into four random groups, one of which received furazolidone 0,04 per cent. for seven days starting 72 hours after infection and another was left untreated as a control. The remaining groups were kept for several hours at room temperature to ascertain the effect of chilling on the severity of the disease. By delaying treatment of one of those lots until deaths occurred it was hoped to compare the value of the drug in circumstances similar to those of a natural outbreak. This proved impracticable through failure of the disease to develop. There were single deaths only in the three untreated groups. All chicks were destroyed for bacteriological examination, three to four weeks later. Although the internal organs appeared normal profuse growths of *S. thompson* were obtained from the liver, spleen and lungs of every one of the 144 untreated chicks and from the intestinal contents of 134. The organism was isolated from the internal organs of only 12 of the 48 treated chicks but it was present in the intestinal contents of 45.

It is difficult to explain the latter findings; reinfection during the interval between cessation of treatment and time of slaughter may have been responsible. The treated pen was in close proximity to three times the number of untreated chicks, all excreting infection. From this and other experiences it would appear that to obtain a true assessment of the efficacy of furazolidone, experimental chicks should be destroyed and examined bacteriologically as soon as treatment is ended. On the other hand, keeping them under observation for a further period may provide a more realistic indication of the value of the drug under natural conditions of chick rearing.

Discussion

The experimental results confirm the value of furazolidone in artificial infections with *S. pullorum* and *S. gallinarum*. At a concentration of 0.02 per cent. in the mash, deaths were prevented during the period of treatment but breakdowns invariably occurred later. A concentration of 0.04 per cent. gave complete protection to chicks against infection with *S. pullorum* sufficiently severe to cause a mortality of 95 per cent. in untreated chicks, and only one chick was later found to be bacteriologically positive. The appearance of the internal organs of the treated chicks was striking; they were completely normal whereas there was extensive nodule formation in the lungs and heart of everyone of the control chicks and those treated with sulphaquinoxaline. This is well illustrated in Figs. 1 and 2.

The effect of the drug on other salmonellae was less definitely determined, but the great reduction in the carrier rate in chicks infected with *S. typhi-murium* suggests that it may play a useful part in controlling this disease. The position of *S. thompson* is similar. The presence of the organism in only 25 per cent. of the treated chicks compared to 100 per cent. of the untreated is suggestive and it would appear that following a diagnosis of salmonellosis a course of furazolidone at a concentration of 0.04 per cent. for 7 to 10 days represents a sound treatment especially in birds intended for the table, such as broilers, turkeys and ducklings.

The ready isolation of *S. thompson* from apparently healthy chicks, as judged by the absence of symptoms of illness during life, and lesions of disease *post-mortem*, raises doubts regarding the significance of the isolation of salmonella from similar chicks received for diagnosis. In such cases the organism would appear to be of low pathogenicity or perhaps even avirulent, and unlikely to be the primary cause of losses.

It is improbable that furazolidone will prove as effective against *S. pullorum* and *S. gallinarum* in the field, for while the weight of infection to which chicks are exposed will generally be lighter (mortality is usually less than 100 per cent. in natural outbreaks), the disease will be more advanced when treatment is begun. The death rate is likely to be higher, and a greater proportion of the surviving birds will probably be carriers. Often the occurrence of deaths is the first indication of the presence of disease in young chicks and there will be a further delay while a diagnosis is obtained. Experiment 5 provides a possible example of what may happen. When treatment was delayed until chicks started to die, deaths continued for a short time but this was followed by rapid control of the disease. The final mortality was just over a third of that of the untreated group in Experiment 4, and more than 30 per cent. of the survivors were excreting the organism. This cannot be regarded as a controlled experiment as there is no means of assessing the weights of infection to which the two groups of chicks were exposed.

The effect of treatment of "carriers" is still uncertain. Gordon and Tucker (1955) reported that at both 0.02 per cent. and 0.04 per cent. fed continuously for 10 days to 13 birds positive to the rapid whole blood test in the field, 11 were subsequently found to be free from *S. pullorum* infection. When the drug was used at 0.04 per cent., seven out of eight birds became negative to the agglutination test 60 to 84 days after the termination of treatment. In preliminary experiments here, with similar birds we have not observed a significant fall in titre and *S. pullorum* has been isolated from some of the treated birds sacrificed for bacteriological examination.

S. pullorum continues to be isolated from a proportion of eggs laid by birds in the control pen but has not been found in the eggs of the treated birds. This work is continuing.

In dealing with the natural outbreaks a concentration of not less than 0.04 per cent. furazolidone should be used and treatment should be continued for 7 to 10 days. Points of great practical importance are, the commencement of treatment at the earliest possible moment and the prevention of reinfection by thorough cleansing and disinfection of brooding equipment and utensils, or the removal of chicks to fresh quarters, immediately after treatment.

Summary

(1) Treatment with furazolidone, 0.02 per cent. in the diet, prevented deaths in groups of young chicks artificially infected with *S. pullorum* and *S. gallinarum*, but breakdowns occurred later. Better results were obtained with double the concentration (0.04 per cent.).

(2) When treatment was delayed until deaths occurred it was less effective but still resulted in a significant reduction in mortality.

(3) Furazolidone was superior to sulphaquinoxaline not only in preventing mortality but in reducing the number of carriers in surviving birds.

(4) Results in infection with *S. typhi-murium* and *S. thompson* were promising but not conclusive.

Acknowledgments.—I wish to express my thanks to my colleagues, Messrs. R. H. Duff, M.R.C.V.S., D. B. Lee, F.I.M.L.T., B. McIntosh, and Miss McDonald, A.I.M.L.T., for their valuable assistance and to Mr. R. C. Hood, F.I.B.P., for the photographs.

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A Septicaemic Disease of Ducklings

BY

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THIS paper presents an account of a disease of growing ducklings which was investigated between July, 1952 and March, 1954.

The disease resembled one which has been described on Long Island, N.Y., by Hendrickson & Hilbert (1932) and in Illinois by Graham *et al.* (1938). A gram-negative pleomorphic bacillus resembling one isolated by the American workers and described under the name *Pfeifferella anatipestifer* was isolated. This organism has been detected in six flocks in Norfolk and Lincolnshire and in addition outbreaks having similar clinical and pathological features have been reported in Surrey, Kent and Staffordshire.

A description of the disease in five of these flocks is given together with an account of some of the characteristics of the organism.

Outbreak L occurred in a flock of 3,000 ducks on an Accredited duck farm of the highest class. The disease first appeared in four-week-old Khaki Campbell ducklings, about 15 per cent. of this group being affected. Mortality during the next two weeks was 5 per cent. At six weeks of age the survivors appeared normal and were indistinguishable from non-affected ducklings in condition, weight and feathering. Losses continued in successive groups at four to six weeks of age and amounted to 30 to 40 ducklings each week. The disease was only seen in Khaki Campbells, none of the Aylesbury ducklings became ill, although they were in intimate contact in the incubators, brooders and growing pens. No complaints were received from the purchasers of the 500 day-old ducklings sold each week.

The most conspicuous symptoms in affected ducklings were staggering inco-ordination and torticollis. A watery discharge from the eyes and nostrils was present in some individuals.

On *post-mortem* examination the most frequent lesions were those of fibrinous pericarditis and peritonitis. Petechiae were sometimes present in the epicardium. The spleen was usually enlarged and mottled with red and grey markings. On cutting, the substance often broke up into greyish seed-like follicles.

Outbreak N occurred on a large duck rearing farm in July, 1952. Losses gradually increased in successive groups to amount to about 30 per cent. by November. In the early stages deaths occurred when the ducklings were three to four weeks of age, but as time passed younger groups became infected. In this outbreak a proportion of the survivors became unthrifty and stunted. In other respects the symptoms were similar to those seen in flock L.

Post-mortem findings resembled those seen in flock L but in addition there was a regular and often massive enlargement of the spleen. In addition to the gram-negative organism described later in this paper an ornithosis-like virus was recovered from affected ducklings.

Outbreak H occurred in a flock of 4,500 growing ducklings; 1,800 laying hens on the premises appeared unaffected. The disease commenced in five-week-old ducklings about one week after they had been moved from the brooders to old loose boxes. Succeeding groups were affected at about the same age. Mortality was up to 75 per cent. in some groups. The owner estimated that about 1,500 ducklings had died in a two-week period. Surviving ducklings showed irregular growth and were disinclined to move about; when stimulated many showed marked inco-ordination. Green discoloration of the excreta was noticeable. Some ducklings had a watery oculo-nasal discharge and a few showed unilateral or bilateral swelling of the hock joints.

A recommendation to replace the ducks' drinking water with a 0·1 per cent. solution of sulphamezathine was misunderstood by the owner, who actually administered a 1·0 per cent. solution. There was a spectacular reduction in mortality within 48 hours. Death rate was reduced to five or six birds a day in the five- and six-week-old groups. There was also a marked improvement in the appearance of the survivors.

Post-mortem lesions were similar to those seen in outbreak L, but there was a greater tendancy to the production of haemorrhages on the serous membranes.

Outbreak R occurred in a flock of 2,000 growing ducklings. Disease was first noted in a group of 200 nine-week-old-ducklings of which 30 died. No further unusual losses occurred until six weeks later when the disease appeared in six-week-old ducklings; mortality amounted to 25 per cent. The disease then spread to five-week-old birds and later to the four-week-old groups. 1,100 laying hens on the farm remained well. Symptoms and lesions in ducklings on this farm were similar to those in outbreak H.

From all four of these outbreaks a gram-negative pleomorphic organism was cultured on sheep blood agar plates streaked with blood, liver or spleen tissue. This organism, however, was not cultured from all specimens examined.

Outbreak O was brought to our attention following a report of suspected Newcastle disease amongst chickens on the premises. This outbreak was subjected to a more detailed examination than earlier ones.

The flock consisted of 800 fowls and 5,700 growing ducklings. On investigation of the disease in the fowls which was later confirmed as Newcastle disease, it was found that there was serious disease amongst the growing ducklings. This disease showed symptoms similar to those observed in the other outbreaks recorded here.

Sera from 20 ducklings were tested for anti-haemagglutinins to Newcastle disease virus. One sample only gave a positive reaction. Two samples showed titres of questionable significance, the remainder were negative.

A consignment of dead and live sick ducklings was obtained for examination. These comprised 21 carcases of ducks which had died immediately before dispatch or in transit and 17 live sick or convalescent ducklings.

Lesions resembled those seen in the other outbreaks and were those of acute septicaemia. The blood was dark in colour and poorly coagulated. The serous membranes showed fibrinous deposits and there was an increased quantity of fluid in the peritoneal cavity and heart sac. This fluid contained flakes of fibrin and was sometimes blood stained. Large blotchy, or petechial haemorrhages were frequently present in the epicardium and heart fat. The lungs and kidneys were congested. The spleen was sometimes enlarged and in most ducklings showed mottling with red and grey markings. On section the spleen broke down into numerous grey seed-like follicles. The hock joints of four of the ducklings were grossly swollen and contused and the joint capsules contained an excessive quantity of turbid viscid fluid.

Cultures were made on sheep blood agar, MacConkey and plain agar plates from liver spleen and heart blood of all ducklings examined and in addition from the heart sac, peritoneum and hock joints depending on the nature of the lesions.

Cultures made from seven of the 21 duck carcases received dead showed heavy overgrowth of miscellaneous organisms on blood plates; of the remaining 14, eight yielded colonies of a gram-negative organism similar to those isolated from the other outbreaks. Three of these isolations were from cultures from the hock joints of four ducklings with joint lesions.

Only one out of five ducklings received alive at the laboratory and examined within 24 hours of arrival yielded a culture of this organism. Three of the nine strains of this organism were tested for pathogenicity by intraperitoneal injection into pairs of growing ducklings. All six ducklings died in two to four days and exhibited typical lesions.

No other organism of significance was isolated. Sera from 15 of the ducklings received at the laboratory alive were all negative for Newcastle disease antihaemagglutinins.

Suspensions of liver and spleen from 17 of the dead ducklings and five of the live sick ducklings were treated with streptomycin and penicillin and injected into the chorioallantoic cavity of embryonated fowl eggs. Neither Newcastle disease virus nor duck hepatitis virus was isolated from any specimen.

Tissue suspensions from 11 of the ducklings but without previous treatment with antibiotics were similarly injected into embryonated fowl eggs. One of these suspensions yielded a minute cocco-bacilliform organism (see footnote in adjoining column).

Spleen suspensions from eight of the ducklings were inoculated intracerebrally into mice. An ornithosis-like virus was demonstrated in the brains of one group of mice. Brain suspensions from these mice were inoculated intraperitoneally into three ducklings. No obvious symptoms were seen in the ducklings, but a slight fibrinous peritonitis with some enlargement of the spleen was present when the ducklings were killed for examination 17 days later.

In a further attempt to determine the cause of this outbreak of disease 11 "survivor" ducklings were inoculated with the virulent gram-negative organism isolated from outbreak N. Two challenge tests were made. In the first test six convalescent ducklings which had been received at the laboratory four days

FOOTNOTE

A suspension of liver and spleen in peptone broth from one duckling of outbreak O, when inoculated into the chorio-allantoic sac of four 10-day embryos without the addition of antibiotic resulted in the death of all embryos in two to six days. The most conspicuous lesion was in the liver which was enlarged, friable and bright pink in colour. Cultures from these embryos on to sheep blood agar plates failed to yield any bacterial growth. Embryonic fluid from these embryos was passed in series through nine passages in embryonated fowl eggs. After inoculation into the chorio-allantoic sac of 10-day-old embryos most died in four to ten days although some were still alive at hatching time. A regular feature in the dead embryos was the swelling and friability of the liver which was usually bright salmon pink in colour but was sometimes mottled and bronze coloured. The spleen was sometimes similarly enlarged. The embryos were stunted and bright pink, oedema was not a feature. Impression smears from the liver and spleen of dead embryos stained by Giemsa and Machiavello methods showed numerous minute cocco-bacillary bodies. These forms were not seen in impression smears from uninoculated embryos. Infective embryonic fluid did not agglutinate R.B.C.'s of chickens or ducks.

The organism grew when inoculated into the yolk sac of 12 six-day-old embryos, all of which died within 12 days.

Inocula treated with streptomycin (500 microgrammes per egg) was not infective whereas similar inocula treated with penicillin (500 units per egg) induced the disease in seven out of 10 embryos.

A filtrate through a 1.02μ APD Gradocol membrane was infective whereas in one test a filtrate through a 0.67μ APD membrane did not infect any of a group of six embryos. Embryonic fluid from the fourth passage was inoculated intraperitoneally into six two-day-old ducklings; four died on the third and fourth days after inoculation and showed enlarged, friable livers. Tissue suspensions from these induced characteristic lesions in chick embryos.

Six two-day-old chicks inoculated at the same time died on the third and fourth days after inoculation and, again, tissue suspensions were infective for chick embryos.

In a larger experiment using two groups each of 50 chicks of 2 and 13 days of age, the mortality was 30 and 14 per cent. respectively, but the growth rate of the survivors was substantially checked.

Mice were resistant to intracerebral and intraperitoneal inoculation of infective embryonic fluids.

This infective agent bears a close resemblance to one which was isolated by Walker & Bannister (1953) from an outbreak of disease in ducks in Canada. This agent was lethal to chick embryos. Swelling and friability of the embryo liver was a notable feature. Unlike the agent reported here, Walker and Bannister stated that their agent agglutinated the erythrocytes of ducks and chickens. This agglutination was not inhibited by Newcastle disease anti-serum. Smears of infective egg membranes revealed small pleomorphic bodies in bacilli, coccoid or pear-shaped forms. Such bodies were not found in normal eggs. Their attempts to reproduce the disease in ducklings, chickens, guinea-pigs, rabbits, rats and mice were unsuccessful.

earlier, together with two susceptible and two immune ducklings of approximately the same age were injected intraperitoneally with a suspension of the 50th subculture of strain N. The two susceptible ducklings died on the third day after inoculation with characteristic symptoms and lesions. The six "test" and the two immune ducklings remained unaffected.

The second challenge test was made with five ducklings from the affected farm, 34 days after their arrival at the laboratory. These ducklings were all believed to have been infected. Two susceptible controls were included. All seven ducklings were challenged by intraperitoneal injection of the 56th subculture of the same strain as the previous test. The two control ducklings died on the second and third day after inoculation. One of the "test" ducklings also died on the third day.

Characteristic lesions were present in all three ducklings and the organism was cultured from blood, liver and spleen.

Characteristics of the Gram-negative Organism

Morphology. A gram-negative pleomorphic organism varying from cocco-bacillary forms to slender bacterial forms having a tendency to slight curvature. Short filaments sometimes formed. Non motile, non sporing.

Cultural Characters. A strict aerobe. Different strains showed considerable variation in rate of growth and colony formation on solid media. On sheep blood agar plates incubated at 38° C. colonies were usually apparent in 48 hours. A poorly growing strain, from outbreak L, sometimes took three to four days before colonies became readily visible. Colonies were mucoid in type, at first pin point with entire edge and smooth glistening surface increasing in size to 1 to 2 m.m. in diameter. Blood not lysed but blood plates may be cleared and browned.

On plain agar growth usually failed, but on Difco peptone water agar, mucoid colonies 1 to 2 mm. in diameter were produced in 48 hours.

In peptone water uniform turbidity with slight deposit evident in 24 to 48 hours. Growth in peptone broth varied from no apparent growth to slight turbidity without deposit. No growth on MacConkey plates.

One strain of organism from each of the five outbreaks was tested in the following peptone water sugars:—glucose, maltose, sucrose, lactose, dulcite, levulose, galactose, salicin, mannitol, inositol, arabinose, sorbite, xylose and rhamnose. No gas was produced by any strain. Two strains produced slight acid in glucose and maltose after seven days. These two strains and one other produced slight acid in galactose.

No change was produced in litmus milk by any strain nor did any produce alkali on litmus milk agar. Indole not produced by any strain.

There was slow liquefaction of gelatine at room temperature by three strains. Gelatine cultures at 38° C. were rendered incapable of solidification after five to seven days incubation. No apparent growth on plain or glycerinated potato in 21 days.

Good confluent growth on Dorset egg and Loeffler's serum in 24 to 48 hours at 38° C. with slow digestion of media on prolonged incubation.

Three strains tested failed to agglutinate with brucella antiserum, nor did these strains produce haemagglutinins for human erythrocytes.

Pathogenicity

Disease could be induced with regularity in ducklings of two to eight weeks of age by intramuscular, intraperitoneal and subcutaneous injection of cultures of all strains. Incubation period with the poorly growing L strain was longer than with the more vigorous strains H and N.

Symptoms in artificially infected ducklings resembled those seen in the natural disease. The first sign of disease was loss of appetite and drowsiness. Incoordination was shown by difficulty in retaining balance. This was most readily observed by watching the bobbing movement of the tail. Spasmodic nodding or shaking movements of the head were common; the bill being often rested on the ground. Leg weakness was progressive. Soon the affected duckling could only move a few paces in a staggering fashion before flopping down. Excreta was moist and coloured green. Ocular discharge was not uncommon. In most inoculated ducklings the disease was rapidly progressive with complete paralysis, coma and death within 24 to 36 hours of the onset of symptoms.

In some ducklings the course was more prolonged with marked nervous symptoms, particularly wry neck and torticollis.

Less severely affected birds made a slow recovery but were frequently unthrifty and undersized.

Several attempts to transmit the disease by pen contact failed but transmission was successful in two of a group of five ducklings dosed by mouth with a pure culture of the organism. In another test three out of five ducklings exposed to an atmosphere containing a pure culture dispersed as a fine mist developed the disease.

The following species have proved refractory to artificial infection:—chicks seven to 14 days old and growing chickens six to eight weeks old by intraperitoneal or intramuscular injection. Geese by intramuscular and intraperitoneal injection. Mice by intracerebral and intraperitoneal injection. Rabbits by intraperitoneal and intramuscular injection. Pigeons by intraperitoneal injection. Guinea-pigs by intraperitoneal and intramuscular injection.

Sensitivity to Antibiotics

In vitro tests showed all strains to be sensitive to penicillin and to a lesser extent to streptomycin, terramycin and chloramphenicol. *In vivo* tests have shown all five strains to be sensitive to sulphamezathine.

The results of four tests on the use of sulphamezathine in artificially infected ducklings are shown in Table I. Sulphamezathine given as a 0·2 per cent. solution replacing drinking water either before inoculation or up to 24 hours after inoculation, effectively prevented the onset of symptoms and all the ducklings tested resisted re-inoculation with the same strain

after the withdrawal of the drug. Sulphamezathine incorporated in the food by mixing dry mash with a 0·25 per cent. solution of sulphamezathine at the rate of one pint to two pounds of mash was also effective. In a further trial in which solutions of 0·2 per cent. and less were used, a proportion of the ducklings sickened and some died.

At the higher levels sulphamezathine was not readily consumed by the ducklings and a check in growth rate was apparent.

Discussion

The evidence that the organism described above is the cause of the disease under investigation is impressive. The 56th subculture on blood agar of Strain N was fully virulent on intraperitoneal inoculation. The organism was regularly cultured from artificially infected ducklings dying within four days of inoculation. A number of artificially infected ducklings either recovered or died after a longer period of illness. From these the organism was only infrequently cultivated. This observation accords with the results of examination of ducklings from outbreak O. Bacteria free filtrates from naturally and artificially infected ducklings were non-infective. The prophylactic effect of sulphamezathine also discounts the theory that a virus might be concerned.

From two of the five outbreaks described in this paper an ornithosis-like virus was isolated. The significance of these isolations was not fully determined but these two strains when inoculated intraperitoneally into growing ducklings did not induce a disease resembling that seen in the field. In fact no symptoms were observed and on *post-mortem* examination lesions were restricted to fibrinous peritonitis and slight enlargement of the spleen.

A further agent of possible significance (see footnote) was isolated from one duckling from outbreak O.

Hendrickson & Hilbert (1932) described a disease of ducks which occurred in Long Island, N.Y., and which presented symptoms and lesions closely resembling those of the present outbreaks.

From this disease they isolated a slender gram-negative bacillus which they called *Pfeifferella anatipestifer*. This organism possessed most of the characters shown by our organism and differed only in minor respects, notably in its inability to act on any of 35 carbohydrates and other fermentable substances and failure to reproduce lethal disease except by intravenous inoculation of cultures into ducklings.

Under the name Duck Septicaemia, Graham *et al.* (1938) described a similar, highly fatal disease in growing ducklings on a farm in Illinois from which they isolated an organism closely resembling that of Hendrickson and Hilbert. This organism resembled ours in that it proved pathogenic to ducklings on intraperitoneal, intravenous and intratracheal injection.

TABLE I
PERIODS OF DOSING SULPHAMEZATHINE AND PENICILLIN

Inoculum	Number of ducklings	Treatment	Result	Re-inoculation of survivors 48 hours after withdrawal of antibiotics
Strain N. 10th subculture	6	Drinking water replaced with 0·2 per cent. solution, sulphamezathine 24 hours before inoculation	No symptoms	No symptoms
" "	6	Penicillin, 2,000 units twice a day intramuscularly (5 days)	"	"
" "	4	No treatment	All died within 3 days of inoculation	
Strain L. 5th subculture	6	Drinking water replaced with 0·2 per cent. solution, sulphamezathine 24 hours before inoculation	No symptoms	No symptoms
" "	6	Penicillin, 2,000 units twice a day, intramuscularly (5 days)	"	"
" "	4	No treatment	2 died within 4 days. 2 developed symptoms and were killed on 5th and 7th days after inoculation	
Strain N. 18th subculture	6	Drinking water replaced by 0·2 per cent. sulphamezathine at the time of inoculation	No symptoms	No symptoms
" "	6	Drinking water replaced by 0·2 per cent. sulphamezathine 24 hours after inoculation.	"	"
" "	8	No treatment	6 died within 4 days. 2 developed symptoms and were killed on 5th day	
Strain H. 5th subculture		Mash prepared with sulphamezathine solution at rate of 1 pint to 2 lb. mash 24 hours after inoculation		
	9	(*a*) Sulphamezathine—1 per cent.	No symptoms	
	9	(*b*) " —0·5 "	"	
	9	(*c*) " —0·25 "	"	
	5	No treatment	5 died within 5 days	

The organism described in this paper also bears a close morphological and biochemical resemblance to that described by Riemer (1904) as being the cause of goose influenza. This organism was termed *Bacillus septicaemiae anserum exudativae* by Riemer but was later placed in the genus *Shigella* under the name *S. septicaemiae* by Bergey *et al.* 1939. The most notable difference between this organism and ours lies in its pathogenicity. Riemer's organism was pathogenic for geese but not for ducks, whereas our organism was regularly pathogenic for ducks but failed to induce symptoms in geese.

Identification of this organism must await further study. It does not appear to fall into any of the usual groups such as *Bacterium*, *Pasteurella*, *Haemophilis vibrio*, etc. It fails to agglutinate with the *Brucella* group sera and did not agglutinate human R.B.C.'s. Absence of growth on bile agar reduces its chances of belonging to the Friedlander's group of organisms despite its mucoid colony. Absence of pathogenicity for mice, pigeons and rabbits and slow liquefaction of gelatine seem to exclude it from the *Pasteurella* group.

Both Hendrickson & Hilbert (1932) and Graham *et al.* (1938) made observations on the use of inactivated cultures for active immunisations. Their results suggested the possibility that such a vaccine would be of material assistance in protecting susceptible birds. We have not attempted to produce a killed vaccine, but in our experiments we observed that inoculated ducklings which have been protected with sulphamezathine were subsequently resistant to re-inoculation. This suggests a possible means of control should circumstances warrant it.

Summary

A gram-negative, pleomorphic bacillus having characters similar to those described in the U.S.A. for an organism named *Pfeifferella analipestifer* was isolated from diseased ducks in six flocks.

Ducklings inoculated intraperitoneally or intramuscularly with cultures of this organism regularly developed a disease similar to that seen in the field.

The onset of symptoms could be prevented by dosing with sulphamezathine.

Ducklings injected with cultures of this organism and dosed with sulphamezathine were resistant to reinfection with the same strain.

An ornithosis-like virus was isolated from ducklings of two of the flocks, and a small, cocco-bacillary organism having pathogenic potentialities was isolated from a duck from one outbreak. Neither of these organisms are believed to have played any important rôle in the outbreaks concerned.

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Warning on the Use of Heating Panels in Small-animal Surgery

BY

R. W. J. KNIGHT and E. M. PITTAWAY

Coventry

IT is common knowledge that general anaesthesia prevents the animal body from regulating and maintaining its body temperature.

There are sound reasons for wishing to control the temperature drop due to surface cooling suffered by animals subjected to considerable surgical interference and trauma. The most important reason is that loss of body heat causes a lower circulating blood volume and haemoconcentration.

" Cold stress " is not completely understood, but causes complex physiological derangements such as increased blood viscosity, hyperglycaemia, eosinopenia and depletion of adrenocortical lipids and ascorbic acid. Histologically one sees fatty and other changes in the liver and kidneys, and marked changes in the adrenal glands. Clinically, we would say that the cold nembutalised dog after severe surgery shows an abnormally high mortality rate and an extremely slow detoxication of the anaesthetic.

Deep Anaesthesia and Temperature Drop

All deep anaesthesia causes a temperature drop which is directly proportional to (1) the duration of time the animal is deeply anaesthetised, (2) the " toxicity " of the dog before operation. This so-called " toxicity " is seen typically in cases of pyometra and is manifested clinically by the inability of the animals to take a normal anaesthetic dose of intravenous barbiturate. Pathological changes usually found in "toxic" animals are dehydration, high blood urea and severe haematological changes, and (3) the degree of trauma inflicted upon the body.

The temperature drop does not vary with different anaesthetics—a point which we have proved with 33 mixed cases of atropine and ether, intravenous nembutal or intravenous thiopentone—but owing to the longer period of deep anaesthesia with intravenous nembutal and its obvious choice for the more prolonged and severe operative procedures, all the biggest temperature drops are found under this anaesthetic.

The rate of temperature drop is presumably varied by the two factors:—

(1) wet or dry sterilisation of operating cloths.
(2) temperature of operating room.

Use of a Heated Panel

In order to control this temperature drop we purchased a heated panel as soon as we were aware that they were on the market. We used it at once with enthusiasm, and set the arbitrary thermostatic control at a heat that felt very warm to the naked hand, but which we thought would be both comfortable and safe to a dog, insulated as it is by hair.

It was found that two large dogs, an Alsatian and a Golden Labrador, both subjected to prolonged surgery, developed extensive lesions seven days after the operation that were clinically identical to a boiling

(*Continued at foot of col. 1, page 859*)

Current Literature

BOOK REVIEW

Experiments in Progress No. 7. 1954-1955. Grassland Research Institute. Price 5s. Cambrian News (Aberystwyth Ltd.).

This issue covers a two-year period.

The institute is now stationed at Hurley, Berkshire, and the task of moving from Drayton nearly completed; from October, this year, Drayton is to become an Experimental Husbandry Farm.

The Director in his prefatory note outlines the work and the investigations undertaken by the various departments; Herbage, Animal, and Ley Agronomy, Biochemistry and Animal Nutrition; Plant Physiology and Statistics. The emphasis is upon team work in which the individual scientist retains a proper perspective of the problem.

Increased attention is paid to the animal, particularly the younger age groups; and some studies concerned with pigs and sheep commence with early pregnancy. Poultry are also included, but only beef cattle.

Improved methods of extraction and fractionation of fresh leafy material are now available for nutritive and pharmacological studies, which are undertaken in collaboration with outside workers, including Weybridge, N.I.R.D. Shinfield, A.R.C., Dept. Biochem., Bangor, King's College, Newcastle, N.I.A.B., Cambridge.

Gordon (Compton) has long advocated the pharmacological study of expressed plant material, this is being done and is surely a great forward step. Experimental work is also undertaken with the N.A.A.S. private owners, and Experimental Husbandry Farms.

This brochure refers to 86 experiments, essentially diverse in character. Increased attention is given to old pasture, including methods of renovation, manurial treatment, pretreatment with herbicides, ecological studies in relation to various grazing treatments, the influence of winter rest, and dung and urine effects. Grazing trials and an examination of micro climatic effects in winter herbage may herald a return to the wider use of foggage.

H24 studies the botanical effects of cattle and sheep grazing on old pastures, and this will surely be published in greater detail. Briefly: severe rotational sheep grazing; a close grass/clover sward with few herbs; severe rotational cattle grazing: an open sward with fairly high proportion of clover and herbs; lenient rotational cattle grazing; a sward of course grass less clover and herbs; range grazing with sheep; a sward of poor grass, little clover and few herbs; range grazing with cattle; a sward of poor grass, little clover and more herbs.

No fertilisers were applied.

Selective grazing by sheep reduced the proportion of *Plantago lanceolata* (Rib grass) and *Chrysanthemum leucanthemum* (Ox-eye daisy).

H15 the pilot grass farm provides a study in the management of different leys to provide grazing for as long a period of the year as possible. The extension of the grazing season by the selection of more persistent and earlier growing grasses embraces more inclement periods, but cases are frequently investigated where the provision of adequate shelter has been overlooked. Food and shelter are both basic requirements for existence, not one without the other. E216 on two simple grass mixtures greater live-weight gains (sig. at 5 per cent. level) were given by lambs grazing rotationally compared with others strip grazing. The recorded carcase weights of the wether lambs (50.8 lb.) were heavier than those of the ewe lambs (46.3 lb.), which is generally the case.

Parasitism in sheep. Studies continue with the comparison of worm-free and infected lambs, and aspects investigated at present are worm-free lambs at pasture, infestation in relation to pasture management, and the level of infestation at which effects are not measurable.

Lung worm infestation of calves and pasture management proceeds in collaboration with Dr. E. L. Taylor (Weybridge). Prior to the introduction of the test animals, infected plots are managed in four different ways: grazed by resistant cattle; by sheep; gang mown; and left to grow.

Post-mortem lung worm counts are undertaken.

The inclusion of lucerne in seeds mixtures for pig grazing from weaning to pork and bacon weight produced greater live-weight gain. These experiments continue with grass or clover dominant swards.

Six different seeds mixtures were used to determine the most suitable for permanent intensively-stocked poultry pens, No. 2 formed the best sward (timothy smooth-stalked meadow grass, yarrow, S184 white clover).

Developments continue in the automatic recording of grazing behaviour; identical twin bullocks are checked against one-minute interval observations—rather exacting work.

The department of ley agronomy deals with the relationship between the grass/legume ley and the soil—its inherent fertility—and this involves root mass and quality, physical condition of soil, microbiology of rhizosphere and root.

The Director expects still greater yields from cereal crops as a result of ley farming.

(Continued from page 858)

water or fat scald. The Alsatian, on which an operation for pyometra was performed, recovered satisfactorily from the operation but lost the skin off its back, from the neck to the tail, to a maximum width of about seven inches; the lesion extended in depth to the subcutaneous fat, some of which was necrotic. The Golden Labrador was operated upon on its side for reconstruction of the pelvis using an external fixation method. In this case the skin was removed from the hind leg and flank of the side of the dog which was lying on the panel.

It is noteworthy that smaller animals subjected to the same heat and similar procedures did not show any skin damage at all, and it is suggested that the weight of heavy dogs flattens the coat and excludes most of the insulating air trapped in the hair. Of course we now use a much lower thermostatic setting, and are able to limit the temperature drop even in extensive procedures to about 1° F. and are delighted with the apparatus.

The value of pasture consumed by the grazing animal is limited by its digestibility. It is rather disappointing to note that the " faecal index " based on regression equations relating herbage digestibility and faecal composition has not proved so accurate. Further work is in progress correlating the digestibility of a wider range of herbage samples with cellulose, fibre, and extracted pigments from oxalated faeces. This again requires further study of faecal production, based on total collection, and the tracer method produced by feeding Cr_2O_3. Contrary to the findings by other workers it has not been possible to establish a fixed pattern of tracer excretion, so that the method of direct sampling from the rectum does not apply; instead the systematic sampling of faeces from fixed circles on the sward is being employed.

Mineral content of plants determined by chromatographic and solvent partition methods is likely to prove as accurate as values obtained by spectrographic methods. Fig. 1, p. 52, outlines a preliminary scheme based on a 10 g. dry-matter sample.

Range figures are also quoted for the following trace elements: Fe., AL., Mn., Cu., Mo., Ni., and Co.

It is interesting to note the wide range quoted for iron, 10 to 250 p.p.m. Possible fluctuation in the total content of this important element tends to be disregarded, perhaps because it is known to form 4 per cent. of the earth's crust. (Values quoted earlier by Mitchell, 50 to 150 p.p.m., were lower than many previously published.)

Extraction of leaf protein fed as a dry concentrate was found inferior to soya bean meal and at present this is not considered economic; its feeding value as a supplement to low protein diet for chicks is limited by low digestibility. Fractionation of plant protein into cytoplasmic and chloroplastic protein, permits further research into methods of disintegrating intact chloroplasts by chemical and physical methods that include ultrasonic vibrations, and this may result in increased digestibility.

It is intended to study the distribution of proteins and amino acids in relation to season and stage of plant growth. The extraction machinery now provides material suitable for pharmacological studies, of sterols, saponins, and growth-depressing factors. In this manner it assists in the collaborative study of bloat grass tetany low S.N.F.

Statistical problems arise, they include the analysis of covariance.

The Director states " there is at present some divergence of viewpoint between agronomists and statisticians. It is necessary for the benefit of field experimentation to bring them into closer partnership."

Whatever the outcome of further biological and allied research, one necessary contribution to the farming community will eventually include management, establishing the greatest measure of positive health and reduced susceptibility, without which increased productivity and economy of production remain difficult to assess or achieve. Among urgent problems that await further investigation that of the successful management of sheep in relation to improved pastures becomes of greater significance with the return of sheep to lowland areas so long dominated by the dairy cow. It is as well not to forget the need for studying the methods of the successful flockmaster, as much as the investigation of the less fortunate flocks: good shepherding was always remarkably unobtrusive, it will not be attained by all, for it requires as well as knowledge a rarer commodity—judgement.

Now that the preliminary work of establishing the Station has been completed, the Director looks forward to preparing a long-term programme of fundamental research. We wish him well!

D. T. M.

DIGEST

Helminth parasites, neoplasia, and encephalitis.

The extent and the nature of the damage caused by certain helminth parasites during their migration through the body, particularly in an intermediate or abnormal host, is the subject of reviews by Schwabe (1955), and Sprent (1955).

The supposition that the severe cellular reaction induced by some endoparasites can assume malignant characteristics is discussed by Schwabe. The nematode, *Gongylonema neoplasticum*, which was believed, as its name implies, to cause neoplasms in the stomach of the rat, is exonerated from blame. The tumours observed were apparently the result of a vitamin A deficiency superimposed upon the helminth infestation—and without any neoplastic tendencies. The author also dismisses, as improbable, a recent report that *Spirocerca lupi*, a nematode which infests the walls of the oesophagus and aortas of the dog, can induce cancer. However, there is no doubt that *Cysticercus fasciolaris*, the intermediate stage of the tapeworm, *Taenia taeniaeformis*, which infests cats, causes neoplasma to develop in the liver of the rat. These tumours, which are usually sarcomata, develop from the fibrous capsule formed by the host around the cysticercus. In cases where rats have been deliberately infected with the eggs of the tapeworm, the neoplasms have formed approximately 250 days after the administration of the eggs. Work is quoted that indicates that the carcinogenetic properties of the cysticercus are associated with its body fluid, although attempts to isolate the active principle by centrifugation and filtration have failed.

The results of an investigation into the various nematode species which gain access to the central nervous system, are contained in reports by Sprent. His work showed that certain of the ascarid larvae invaded nervous tissue when the embryonated eggs were ingested by an abnormal host, although this intra-cranial migration was not apparent when the natural host was infected. Experiments with mice indicated that the larvae of the dog ascarid *Toxocara canis*, were frequent invaders of the brain, and that they remained in this region for several months, apparently (in the mouse), without causing nervous symptoms. However, reports are quoted of the recovery of ascaris larvae from the brains of children, which suggests that the invasion of the central nervous system by *T. canis* may not be confined to the rodent.

D. W. J.

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News and Comment

THE ROYAL COLLEGE OF VETERINARY SURGEONS

The following ladies and gentlemen were admitted to membership of the Royal College of Veterinary Surgeons on the date shown against their entries:—

Barker, David Julius. Oct. 14, 1955. Amberley Court Lodge, Amberley, Stroud, Glos. B.V.SC. (Bristol).

Baxter, Mary Katherine (Miss). Oct. 14, 1955. 39, Clatterford Road, Carisbrooke, Isle of Wight. B.V.SC. (Bristol).

Bracewell, Colin Derek. Oct. 14, 1955. 20, Windmill Avenue, Ewell, Surrey. B.V.SC. (Bristol), B.SC. (Zool.), (London).

Carter, George Robert. Oct. 14, 1955. 21, Glen Park, St. George, Bristol, 5. B.V.SC. (Bristol).

Evkersley, James Miles. Oct. 14, 1955. 4, Leamington Grove, Swindon, Wilts. B.V.SC. (Bristol).

Field, Charles Michael Johnson. Oct. 14, 1955. Newton House, Cross, Nr. Axbridge, Somerset. B.V.SC. (Bristol).

Hardern, Ernest John Dranfield. Oct. 14, 1955. Castle Farm, Old Town, St. Mary's, Isles of Scilly. B.V.SC. (Bristol).

Howe, Michael Lainson. Oct. 14, 1955. 73, Northlands Road, Southampton, Hants. B.V.SC. (Bristol).

Johnson, Anthony Withall. Oct. 14, 1955. Tanglewood, Scotts Lane, Shortlands, Kent. B.V.SC. (Bristol).

Keen, Peter Marley. Oct. 14, 1955. 21, Bath Road, Melksham, Wilts. B.V.SC. (Bristol).

Mann, John Cyril. Oct. 14, 1955. 10, David Place, St. Helier, Jersey, Channel Islands. B.V.SC. (Bristol).

Marriott, Philippa Karen (Miss). Oct. 14, 1955. Whistlers, South Petherton, Somerset. B.V.SC. (Bristol).

Robinson, Derek John. Oct. 14, 1955. 42, Norton Road, West Cross, Swansea, Glam. B.V.SC. (Bristol).

Snow, Michael Keith. Oct. 14, 1955. 185, Canterbury Street, Gillingham, Kent. B.V.SC. (Bristol).

Spark, David John. Oct. 14, 1955. 22, Garfield Avenue, Dorchester, Dorset. B.V.SC. (Bristol).

Spranger, Jane Phoebe Costanza (Miss). Oct. 14, 1955. Old Malthouse, Ashford Hill, Nr. Newbury, Berks. B.V.SC. (Bristol).

Vowles, Graham Bowater. Oct. 14, 1955. Neathwood, Kingswood, Wotton-under-Edge, Glos. B.V.SC. (Bristol).

Williams, Dewi George. Oct. 14, 1955. Glennydd, Hendy Road, Mold, Flints. B.V.SC. (Bristol).

Wright, John Derek Edwards. Oct. 14, 1955. Windrush Cottage, Castle Hill, Banwell, Somerset. B.V.SC. (Bristol).

THE REGISTER OF VETERINARY SURGEONS

Under Section 25 (3) of the Veterinary Surgeons Act, 1948, and the provisions of Statutory Instrument No. 1150 of 1953, the names of the following gentlemen have been removed from the Register of Veterinary Surgeons. * Indicates those who have requested the removal of their names from the Register.

*Berry, E., Greenhill, Leckhampton Hill, Cheltenham.

Coe, T., 14, Broombill Road, Belfast.

Evans, T. M., Wynston, Church Street, Caernarvon.

Galloway, J. D., c/o 55, Shakespeare Road, Bedford.

*Godwin, G. W., c/o Grindlay & Co., 54, Parliament Street, S.W.1.

Harris, P. J., c/o Glyn, Mills & Co., 22, Whitehall, S.W.1.

*Hobson, J., Carrig Lodge, Inverness, Nova Scotia, Canada.

*Holland, A. B., Stretton Road, Much Wenlock, Salop.

*Kingcombe, M. H., Pavlova Farm, P.O. Umvuma, S. Rhodesia.

*Moir, G., 81, Ashley Road, Aberdeen.

*Nixon, H. B., Hill View, Tadley, Nr. Basingstoke, Hants.

*Panton, A., Dalgreine, Blair Atholl, Perthshire.

*Rowbotham, S. C., Ashlay Lodge, Upper Parkstone, Dorset.

*Smith, R. T., c/o Lloyds Bank, Melton Mowbray, Leics.

*Thompson, P. R., 43, West Street, Oundle, Northants.

The following names have been removed from the Supplementary Veterinary Register:—

*Keech, T., Wynford, Halstock Leigh, Yeovil, Somerset.

*Wake, S. F., 390, Wootton Road, King's Lynn, Norfolk.

R.C.V.S. LIBRARY ACCESSIONS

Books

ANTHONY, D. J. Diseases of the pig and its husbandry. *Fourth edition.* London, Baillière, Tindall, & Cox, 1955.

BOYD, H. Bovine genital vibriosis. A large-scale field investigation, using the vaginal mucus agglutination test. Uppsala, Almquist & Wilsell, 1955.

BURNET, F. M. Principles of animal virology. New York, Academic Press, 1955.

DUKES, H. H. The physiology of domestic animals, with a chapter on physicochemical basis of physiological phenomena, a section on physiological oxidations, a chapter on water, electrolytes, and acid-base balance, a part on intermediary metabolism and a part on endocrine organs, reproduction and growth. *Seventh edition.* London, Baillière, Tindall & Cox, 1955.

GREAT BRITAIN. *Medical Research Council.* The diagnosis and treatment of haemophilia and its related conditions. R. G. Macfarlane & Rosemary Biggs. London, H.M.S.O., 1955.

Reports

AUSTRALIA. *Commonwealth Scientific and Industrial Research Organisation.* Sixth Annual Report for the year ending June 30th, 1954.

EAST AFRICA HIGH COMMISSION *East African Tsetse and Trypanosomiasis Research and Reclamation Organisation.* Annual Report, 1954-55.

EIRE. *Department of Agriculture.* Twenty-third Annual Report of the Minister for Agriculture, 1953-54.

GREAT BRITAIN. *Ministry of Agriculture, Fisheries and Food.* Report on the Animal Health Services in Great Britain, 1954-55.

HILLSBOROUGH. *Agricultural Research Institute of Northern Ireland.* Twenty-eighth Annual Report, 1954-55.

JAMAICA. *Department of Agriculture.* Annual Report, 1953.

JAMAICA. *Department of Agriculture Investigations.* 1953. (Bull. 53. N.S.)

LONDON. *National Central Library.* Thirty-ninth Annual Report for the year ending February, 1955.

MADRAS *Veterinary College.* Annual Golden Jubilee Commemoration Volume, March, 1955.

MADRAS *Veterinary College.* Calendar for the Academic Year, 1955-56.

MUKTESWAR. *Imperial Veterinary Research Institute, Mukteswar and Izatnagar.* Report, 1946-47.

NEW ZEALAND. *Department of Agriculture.* Annual Report for the year ending March 31st, 1955.

NEW ZEALAND. *Department of Scientific and Industrial Research.* Twenty-ninth Annual Report, 1954-55.

NORTH BORNEO. *Department of Agriculture.* Annual Report, 1954. 1955.

ZANZIBAR PROTECTORATE. *Department of Agriculture.* Annual Report, 1954.

JOURNAL OF THE A.V.M.A.

As from January, 1956, the Journal of the American Veterinary Medical Association, which has been published monthly for over 75 years, will become a twice monthly publication. It will be dated the 1st and 15th of each month.

We are informed that among the reasons for this change are the steadily increasing demands upon the journal from both authors and advertisers for more space for publication of acceptable material, the desire of the Association and its editorial staff to present more worthwhile articles in the fields of special interest to veterinary surgeons, such as small-animal medicine and surgery, and the earlier publication of manuscripts, thereby reducing the current backlog of papers. The price of the journal will be increased as present annual subscription expires. After December 31st, this year, the subscription for overseas readers will be $16.50.

In wishing our contemporary every success, and welcoming the greater frequency of its publication, we may note that THE RECORD has recently been called upon to face similar problems. As announced in a recent Supplement a substantial additional annual sum has been voted to the Editorial Committee for the further expansion and improvement of our own journal, one result of which will be more speedy publication of original work.

FOWL PEST ORDER

During the last few weeks about 30 outbreaks of fowl pest have been confirmed on premises in central Norfolk. With a view to bringing the situation under more effective control, the Minister of Agriculture, Fisheries and Food and the Secretary of State for Scotland have made an Order entitled The Live Poultry (Mid Norfolk) Order, 1955. The main effect of this Order is to prohibit as from November 9th the movement of live poultry (except day-old chicks and hatching eggs) into, out of and within the area specified below:—

Scheduled Area

The City and County Borough of Norwich.
The County Borough of Great Yarmouth.

In the County of Norfolk

The Petty Sessional Divisions of Blofield and Walsham, Depwade, Diss, Earsham, East and West Flegg, Eynsford, Forehoe, Guiltcross and Shropham, Loddon and Clavering, Mitford and Launditch, Taverham and Wayland.

In the Petty Sessional Division of South Greenhoe

The parishes of Necton, Holme Hale and Bradenham.

In the County of East Suffolk (Petty Sessional Division of Mutford and Lothingland)

The parishes of Burgh Castle and Bradwell.

PERSONAL

Births

HANDS.—On October 30th, 1955, at Birmingham Maternity Hospital, to Joan, wife of R. R. Hands, B.SC., M.R.C.V.S., 158, Birmingham Road, Sutton Coldfield, Warwicks, a son, Christopher Roy.

MURCH.—On November 4th, 1955, at St. Andrews, Melbourne, to Anne, wife of Oliver Murch, B.V.SC., M.R.C.V.S., of 32, Stirling Road, Croydon, Victoria, Australia, a daughter.

Marriage

PALMER—HAYNES.—At St. Andrew's Church, Lammas, on November 5th, Anthony Claude Palmer, B.A., M.R.C.V.S., of Swannington Hall, Norwich, to Shirley Marilyn Haynes of Toronto.

COMING EVENTS

November

15th (Tues.). General Meeting of the South-east Midlands Division, B.V.A., at Franklin's Garden Hotel, Northampton, 8 p.m.

16th (Wed.). General Meeting of the Supplementary Veterinary Register Division, B.V.A., at 7, Mansfield Street, Portland Place, W.1, at 2.30 p.m.

17th (Thurs.). Annual Dance of the Victoria Veterinary Benevolent Fund, County of Ayr, Ladies Guild at Western House, Ayr.

General Meeting of the Midland Counties Division, B.V.A., at the Birmingham Medical Institute, 154, Great Charles Street, Birmingham, 2.30 p.m.

General Meeting of the South Eastern Division, B.V.A., at the Royal Star Hotel, Maidstone, 2.30 p.m.

General Meeting of the Herts and Beds Division, B.V.A, at the Red Lion Hotel, Luton, 7.30 p.m.

18th (Fri.). Annual General Meeting of the Scottish Branch, B.V.A., in the Royal (Dick) School of Veterinary Studies, Edinburgh, 2.30 p.m.

24th (Thurs.). Autumn Meeting of the Eastern Counties Division, B.V.A., at the Bedford Lodge Hotel, Newmarket, 12 noon.

Meeting of the Essex Veterinary Division, B.V.A., at the Essex Institute of Agriculture, Writtle, Nr. Chelmsford, 7.30 p.m.

25th (Fri.). Ordinary General Meeting of the Royal Counties Division, B.V.A., at the Caversham Bridge Hotel, Reading, 2.30 p.m.

30th (Wed.). Meeting of the Southern Counties Division, B.V.A., at Salisbury, 7.30 p.m.

December

1st (Thurs.). General Meeting of the Central Division, B.V.A., at the Royal Veterinary College, Camden Town, N.W.1, 6 p.m.

5th (Mon.). Annual Dinner of the Centaur Society, University of Bristol, at the Hawthorn's Hotel, Bristol, 7 p.m.

R.C.V.S. EXAMINATIONS

December 8th, Thursday.—M.R.C.V.S. Written Examinations.

December 9th, Friday.—M.R.C.V.S. Written Examinations.

December 12th, Monday.—M.R.C.V.S. Practical and Orals commence (Panels " A " and " B ").

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ayr. Walston Farm, Fenwick, Kilmarnock (Nov. 4).

Ches. Coronation Farm, Delamere, Northwich (Nov. 5); Daisy Bank Farm, Sproston, Holmes Chapel, Crewe (Nov. 7).

Denbigh. Lodge Farm, Llangynhafal, Denbigh (Nov. 2); Blackwood Farm, Penley, Wrexham (Nov. 7).

Derby. Crowtrees Farm, Shottle (Nov. 1); Pasture Tops Farm, Fenny Bentley, Ashbourne (Nov. 2); Gorse Hill Farm, Parwich, Ashbourne (Nov. 4); Clives Farm, Mugginton (Nov. 5).

Dumfries. Conrick Farm, Sanquhar (Nov. 1).

Flints. Stamford Dairy Farm, Holywell; New Hall Farm, Willington (Nov. 2); Gwern-y-Ffynnon, Rhosesmor, Mold (Nov. 7).

Lancs. Head Nook Farm, Myerscough, Garstang, Preston (Nov. 1); Bond Gate Farm, Dolphinholme, Lancaster (Nov. 3); Chilsey Green Farm, Stoneyhurst, Blackburn (Nov. 5); College Farm, Upholland, Wigan (Nov. 7).

Montgomery. Bank Farm, Bulthy, Middletown, Welshpool (Nov. 7).

Northampton. Grange Farm, Earls Barton (Nov. 4).

Roxburgh. Crailing Home Farm, Crailing, Jedburgh (Nov. 5).

Salop. Green Farm, Longnor, Shrewsbury (Nov. 1).

Staffs. Moss Rose Farm, Moddershall, Stone (Nov. 2); Odda Hall Farm, Ipstones, Stoke-on-Trent; Woodhouse Farm, Wood Lane, Bignall End, Stoke-on-Trent (Nov. 3); Farbrook Farm, Quarnford (Nov. 4); The Mount, Adbaston (Nov. 7).

Westmorland. Dallan Bank, Great Strickland (Nov. 5).

Yorks. West End Farm, Castle Bolton, Leyburn; East Borwins Farm, Borwins, Bainbridge; Thwaite Holme Farm, Askrigg, Leyburn (Nov. 2); Tofts Farm, Pickering (Nov. 3); Novia Farm, Carlton, Yeadon, Leeds (Nov. 4); Cold Harbour Farm, Yeadon, Leeds (Nov. 5); Halfway House, Leeds Road, Selby (Nov. 7).

Fowl Pest

Essex. Ramblers Cottage, Dawes Heath, Hadleigh (Nov. 2); Studio, Long Road, Canvey Island (Nov. 4); Tylerset Farm, Dawes Heath, Hadleigh (Nov. 7).

Lancs. The Hut, Hoghton, Preston (Nov. 1); Chestnut Farm, Halshall, Ormskirk; Edge End Field, Stanworth, Withnell, Chorley (Nov. 2); White House Farm, Hall Lane, Longton; Moss House Farm, Little Hoole, Preston (Nov. 3); Whalebone Farm, Marsh Lane, Longton; Middle Grange Farm, Grange Lane, Hutton, Preston (Nov. 5); Longcroft, Gill Lane, Longton, Preston (Nov. 7).

Norfolk 3, Council Houses, Great Moulton, Norwich (Nov. 1); 2, Clint Green, Yaxham, East Dereham (Nov. 3); King Street, New Buckenham, Norwich (Nov. 4).

Yorks. Glenview Farm, Wheatley, Halifax (Nov. 1).

Swine Fever

Ayr. Hill Farm, Loudoun, Galston (Nov. 2).

Cambs. Meadowhurst, East End, Isleham (Nov. 8).

Devon. Teignbridge House, Kings Steignton, Newton Abbot (Nov. 5).

Dorset. Hiscocks Farm, Plush, Piddletrenthide, Dorchester (Nov. 8).

Essex. Five Acres, Burches Road, Thundersley (Nov. 3).

Gloucs. Spoonbed Farm, Painswick, Stroud (Nov. 3); Feofee Farm, Itchington, Tytherington (Nov. 8).

Lanarks. Slakiewood Cottage, Muirhead, Glasgow Nov. 5).

Lancs. The Mount, Waterworks Lane, Winwick, Warrington (Nov. 2); Boggart House Farm, High Moor Lane, Wrightington, Wigan (Nov. 3).

Norfolk. Chapel Farm, Salters Lode, Downham Market (Nov. 2); Field 13, Shrub Hill Farm, Feltwell, Thetford (Nov. 3); Glenville House, Barroway Drove, Stow Nordelph, Kings Lynn (Nov. 8).

Northants. Nissen Hut in Broadway Field, Dodford Lane, Norton, Daventry (Nov. 8).

Somerset. Lime Kiln, Maidenbeech, Crewkerne (Nov. 7).

Staffs. Piggeries, rear of Foxes Row, Watling Street, Brownhills, Walsall (Nov. 3); Coton Lane, Hopwas, Tamworth (Nov. 7).

Stirling. Low Blochairn Farm, Baldernock; Laurel Hill Nursery, Laurel Hill Place (Nov. 7).

Warwicks. Broad Marston Fields Farm, Long Marston, Stratford-on-Avon (Nov. 4); Post Office Stores, Bishops Itchington, Leamington Spa (Nov. 8).

Yorks. Headfield Farm, Saville Town, Dewsbury (Nov. 8).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

THE PROFESSION AND THE PRESS

Sir,—Recent issues of a leading national farming newspaper have contained letters from farmers complaining of the price of antibiotics supplied to them by veterinary surgeons. Later came complaints of veterinary surgeons' charges for all services, closely followed by an editorial on the first page in which almost every department of our profession was criticised.

The next edition of this journal contained quite a long article about veterinary surgeons' drug charges, in which were quoted extracts from the confidential letter on this subject by the B.V.A. not long ago. Also quoted were extracts from the meeting of the Society of Practising Veterinary Surgeons, in which one of their members is reported to have said that high drug charges will " kill the goose that laid the golden eggs." The death of this goose would be welcomed by all if the dreadful cliché died with it, but it is not a joke to see our own confidential veterinary matters quoted back at us by the farming Press, especially in this context.

The most disappointing aspect of this matter, however, is the very poor response from official quarters to this attitude of suspicion and antagonism by the farming Press. The B.V.A. letter in reply missed the next issue of the paper, due, I believe, to the fact that nothing was known about the situation early enough for anything to have been done about it. When the B.V.A. letter was printed a fortnight later it was weak and in parts irrelevant. It started with the unbelievable statement that the " B.V.A. welcomes the general tenor of the leading article," etc. The only other reply was by a practitioner who signed himself " Veterinary Surgeon," and whose letter was constructive and fair. The editor's note made at the foot of this courteous and restrained letter by " Veterinary Surgeon " was a remarkable exhibition of bad manners, so much so that one's anger quickly turned to wonder that the editor of a presumably responsible paper should allow such things to be printed. From these events emerges the fact that the efficiency of the liaison between our profession and the Press has reached a record low level.

Many of our members may be against entering into correspondence in newspapers, yet it is often these same members who complain of the low status of our profession and of the lack of publicity which prevents us taking a higher place in the community.

In *The Times* recently a letter appeared from the secretary of an animal protection society, in which he stated that cruelty to animals by children was the most common form of child crime to-day. A magistrate who dealt with juvenile delinquents replied by saying that in 19 years he could remember only one case. Several letters followed. The subject may or may not be important but here was a case in which a round letter from the official sources of our profession would at least have shown that we were interested in the problem. Are we not supposed to be experts where animal health is concerned? Nowhere in this correspondence were the words " veterinary surgeon " printed, and it is doubtful whether any of the writers ever thought about our profession in connection with this subject. The animal protection societies with branches North, South, East and West were much lauded by independent authors and by themselves.

It is time that a first-class Press liaison was organised by us which should be accorded the high position in the profession which its importance deserves.

Yours faithfully,

A. KIRKWOOD.
J. MEIKLE.

14 & 16, Queen's Road,
Reading.

November 8th, 1955.

JOHNE'S DISEASE IN A LAMB

Sir,—Johne's disease is essentially a disease of adult sheep. The clinical picture is rarely seen in animals less than two years old but that the disease can occur in lambs is shown by what follows. One of us (M.H.C.) conducted a *post-mortem* examination upon a six-month-old lamb which had a history of loss of condition and severe scouring before death. A tentative diagnosis of parasitic gastro-enteritis was made and the abomasum and intestines were despatched to the Veterinary Investigation Centre at Aberystwyth with a request that the material be examined for parasites. During the past year in the flock under investigation some 10 to 12 animals, all lambs, had died at fairly regular intervals after losing condition and scouring from about three weeks. The remainder of the flock at the time of the *post-mortem* examination was in excellent condition and showing no evidence of diarrhoea. A large selection of anthelmintics including phenothiazine, arsenic and tetrachloroethylene had been used without any apparent good effect.

A laboratory examination of the *post-mortem* material from the lambs revealed necrotic areas in the lymph nodes, some of which contained inspissated or fluid pus. Smears prepared from these sites revealed the presence of innumerable acid-fast bacilli indistinguishable morphologically from *M. paratuberculosis*. Smears from the bowel mucosa also contained large numbers of similar organisms although the mucous membrane itself appeared relatively normal except for a greenish discoloration. Sections prepared from the lymph nodes confirmed the previous

findings in that they, too, contained large numbers of organisms resembling *M. paratuberculosis*.

Unfortunately no *post-mortem* examinations were made on the lambs which had died earlier, but the owner of the animals believed that they had died from the same cause. On the other hand it is possible that *Nematodirus* was responsible as the history and the symptoms are similar to those present in a gastro-enteritis caused by these nematodes. Losses from *Nematodirus* in lambs have been heavy this year throughout Mid-Wales.

Yours faithfully,

M. H. CROSS,
L. E. HUGHES.

Ministry of Agriculture,
Veterinary Investigation Centre,
Dairy Buildings,
Llanbadarn Road,
Aberystwyth.

November 1st, 1955.

ASSISTANTS IN VETERINARY PRACTICE

Sir,—I am indebted to Mr. Muir for so kindly clarifying my "confusion of thought." I am confident that my power of self expression will improve, now that I have such a worthy critic. It is only after a number of years of submission to my seniors that I have at last found a practice in which I am able to regain a measure of the self confidence I had on my graduation day—confidence that was almost shattered by the exacting behaviour of some of those with whom I served my apprenticeship.

I am not so concerned with the problems of wealthy assistants—they are a fortunate minority. I am concerned with the problems facing assistants of average means, in particular that of future establishment. I know that the question of future establishment of veterinary practices in this country receives the attention of committee members of the B.V.A. from time to time; but may I suggest that a representative body of assistants, who are so vitally concerned, could make a valuable contribution towards solving the problems involved?

Applications for assistants in the advertisement columns of THE VETERINARY RECORD frequently include the words "capable of taking sole charge in absence of the principal." Surely if an assistant fails to assume responsibility during his principal's absence from practice he is not competent to hold his position; and while no practice may have its receipts enhanced by the absence of the principal, does not that also apply with equal force in the case of absence of the assistant?

Whether or not matrimonial bliss helps a young graduate on the threshold of a professional career is a matter of opinion. Mr. Muir suggests it is sufficient compensation for the hardships of the new graduate during his first few years in general practice. That hardship used to exist is unfortunately true; but in these more prosperous days, when qualified assistants are at a premium, unjust conditions of employment are not accepted for long.

I agree that I am dependent on my own skill and efficiency to secure and keep a job. However, as a professional man I make a distinction between having a job and pursuing a vocation. In addition I require the opportunity to develop my skill and efficiency, and freedom to live as an individual.

The day has almost passed, I hope, when assistants are treated as necessary encumbrances. Nowadays, their professional status, at any rate, is recognised by the new generation of principals. Perhaps this will be sufficient explanation for my use of the colloquialism " coming into their own."

Yours faithfully,

JOHN FIELD.

15, Jubilee Avenue,
Grantham,
Lincolnshire.

November 2nd, 1955.

THE OPENING OF CONGRESS

Sir,—I would put no impediment in the way of those who prefer to make their private devotions in public, but your correspondent who hurls at his fellow men the phrase "the choking foulness of the modern world" obviously stands in need of something more. To widen his horizon I suggest a course of Robert Browning's poems, with "Paracelsus" as a start.

Yours faithfully,

ESMOND BROWN.

Lan House,
Weston Road,
Bath.

November 6th, 1955.

VETERINARY APPLICATION OF 5:7-DICHLORO-8-HYDROXYQUINALDINE 3 PER CENT.*

Sir,—The above preparation, in the form of a non-greasy ointment, has been used extensively on small-animal patients over the past year, after reports from doctors of its efficacy in dealing with skin lesions on their own pets.

It is an antiseptic and antimycotic dressing which is pleasant for owners to use. Its efficiency seems to be little impaired by persistent licking, nor have there been any reports of side effects.

While it has been used as a dressing for skin lesions, wounds, burns, etc. of all kinds, it has proved most useful for the treatment of moist eczema. Two typical cases are reported:—

(1) *Six-year-old Boxer bitch* with acute moist eczema extending over the whole of the chest and brisket. This bitch was first seen after it had been treated for some time and the owner was considering euthanasia. The bitch was admitted to hospital and dressed with crystal violet jelly—with no improvement. The ointment was then used twice daily and the patient was discharged four days later—the whole area being dry and well on the way to recovery.

(2) *Two-and-a-half-year-old male cat* (neutered) with extensive areas of moist eczema on abdomen

* Trial supplies of 5:7-Dichloro-8-hydroxyquinaldine 3 per cent. (Steroxin Ointment) were obtained from Geigy Pharmaceutical Company Ltd., Manchester.

and thighs resulting from persistent licking of miliary eczema. Various astringent dressings which were applied had no effect beyond making the patient sick. The ointment was then used twice daily and, coupled with a testosterone implant, effected an almost complete cure. Occasional slight recurrences are kept under control by the owner using this ointment.

Yours faithfully,

ARTHUR G. WELCH.

4, Robertson Road,
Brighton, 5,
Sussex.

November 8th, 1955.

FLUORINE POISONING ARISING FROM INDUSTRIAL CONTAMINATION

Sir,—In his review "Fluorine Alleviators" (VETERINARY RECORD, **67,** 827) Professor Boddie gives references to the above, but fails to include what I believe to be the first record in Britain—appearing in my name—in the 1937 Annual Report of the North of Scotland College of Agriculture, Aberdeen.

The subjects were sheep grazing in the vicinity of an aluminium works in Inverness-shire. They were owned by a Mr. Cameron—a fact that may explain Professor Boddie's mention of that name.

Yours faithfully,

JOHN C. GRANT.

Veterinary Department,
Marischal College,
Aberdeen.

November 2nd. 1955.

THE CLUB SYSTEM IN NEW ZEALAND

Sir,—We have followed with great interest the correspondence concerning club practice in New Zealand. We have both had several years experience of private practice in Britain and also in the club system here.

It must be admitted that there are imperfections within the club system, but does this not also apply to a system of private practice? We certainly think so, having had some experience of both.

It is alleged by some that the standing of the profession is lowered by the club system. We suggest that the status of the profession, of any profession, is the result of the competence and bearing of the individual members, and does not depend on the system of practice within which they work.

With regard to standard of living, we have found that we enjoy this on a much higher scale than we did in Britain. We also suggest that, generally speaking, the club veterinary surgeon's living standards are higher than those of veterinary surgeons employed as assistants in private practice in Britain.

We feel also with regard to Mr. Williamson's statement in his letter of June 27th, 1955, concerning the " forseeable rate of remuneration after the five years from graduation " that the ancient saying " as ye sow, so shall ye reap " applies much more so in the club system here than it does to assistants in practices in the United Kingdom.

Yours faithfully,

E. L. OWENS.

Box 220,
Hamilton,
New Zealand.

HUGH WARNOCK.

Box 21,
Morrinsville,
New Zealand.

October 21st, 1955.

TICKBORNE FEVER IN DAIRY CATTLE

Sir,—We have recently encountered an outbreak of tickborne fever in a herd of dairy cattle in this part of the country. As there appears to be few references to the subject of the disease in cattle we thought that a note on our observations might be of interest.

A herd of Shorthorns was moved last winter from one area to a new farm. On this particular farm, ticks were present and a few cases of redwater had been encountered in the past. During the herd's first summer there were a few cases of an indefinite illness, characterised by a sudden drop in milk yield and a loss of appetite. The animals recovered in two or three days without treatment and no advice was sought at this time. In October this year there were several cases, and some of these did not return to their normal yield and were off their food for as long as a week. On examination, there was little to find except a high temperature up to 106° F.

As it was not possible at this first visit to see or obtain a sample of urine, blood smears were taken to see if babesiasis was present. These were negative and further blood samples were taken. At this second visit, the urine was seen to be normal in colour. The blood samples proved positive to rickettsia. As regards treatment, it would appear that only the broad spectrum antibiotics would be effective and it was felt that owing to their cost, their use should be restricted to severe cases. As yet, we have not used them. As a palliative, febrifuge has been prescribed, but we should be pleased to hear of the experiences of any of our colleagues. We presume that this herd will become "salted" in time (if one may use this expression) and that the disease will become self-limiting.

It would appear that the disease in cattle can cause definite clinical signs and be a source of financial loss to the farmer, as in some cases the cow does not return to the full yield which it was giving before the disease occurred.

Yours faithfully,

J. B. TUTT,
C. LOVING.

1, St. Cross Road,
Winchester.

October 21st, 1955.